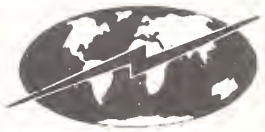
# Foreign Animal Disease Report

United States
Department of Agriculture

Animal and Plant
Health Inspection Service

Veterinary Services

Emergency Programs



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## This Issue





## Emergency Programs Activities

**Field Investigations.** During the second and third quarters of fiscal year 1995 (January 1, 1995–June 30, 1995), veterinary medical officers from the U.S. Department of Agriculture, Animal and Plant Health Inspection Service, Veterinary Services (USDA, APHIS, VS), and State departments of agriculture conducted 88 investigations of suspicious foreign animal diseases in the United States to eliminate the possibility that an exotic disease may have been introduced. These investigations included 34 (40 percent) for vesicular disease conditions, 18 (20 percent) for bovine spongiform encephalopathy surveillance, 13 (15 percent) for encephalitic disease, 13 (15 percent) for avian diseases in pet birds and poultry, 3 (3 percent) for hemorrhagic septicemia, 1 (1 percent) for mucosal disease, 1 (1 percent) for spontaneous abortion, 1 (1 percent) for excessive, acute death, 1 (1 percent) for myiasis/acariasis, 1 (1 percent) for pox/lumpy skin disease, and 2 (2 percent) for other miscellaneous disease conditions.

There were 24 investigations conducted in VS' Northern Region, 8 in the Southeastern Region, 10 in the Central Region, and 46 in the Western Region. All investigations were negative for foreign animal diseases or pests.

## Foreign Animal Disease Diagnostic Laboratory (FADDL) Activities

**Laboratory Relocation.** The National Veterinary Services Laboratories' (NVSL) FADDL, located on Plum Island, NY, is moving after more than 40 years of continuous service as the primary exotic animal disease diagnostic laboratory in the United States. The old laboratory, housed in Building 257 on the island, is closing. The entire laboratory has been moved across the island to newly renovated laboratories in Building 101. This move is the culmination of

nearly 10 years of planning to consolidate all of the island's operations to a larger research laboratory and surrounding buildings. The new laboratory includes approximately 24,000 square feet of space.

The recent outbreak of vesicular stomatitis virus in the Western United States occurred during the middle of the move. Despite the arrival everyday of multiple high-priority laboratory specimens related to the outbreak, the move was accomplished without interruption of diagnostic services. This achievement is a credit to the careful planning of the laboratory staff who coordinated the move.

It is a complex operation to move an entire high-containment laboratory without shutting down operations. Each item of equipment had to be sealed in two layers of 10-ml-thick, heat-sealable, shrink-wrap plastic material before being decontaminated, removed from the laboratory, and transported to the receiving airlock in the new building. Small equipment and reagents were transported in 4-foot-square, stainless-steel, airtight containers. Even a 3/4-ton electron microscope was wrapped and transported without mishap.

The final stage of decommissioning and mothballing Building 257 will now proceed. This step will require total decontamination of the inside of the building with formaldehyde gas. The building will then be sealed for an indefinite period of time. There are no plans to reopen the building at this time.

**Retirement.** In July 1995, Dr. Charles Mebus retired from his position as laboratory chief of NVSL–FADDL. Mebus had been laboratory chief since 1988 and had served as research leader of the pathobiology section in USDA's Agricultural Research Service (ARS) laboratory on Plum Island since 1977.

Mebus received his doctor of veterinary medicine degree from Cornell University in 1956 and his doctor of philosophy degree from Kansas State University in 1963. He is a diplomate of the American College of Veterinary Pathologists and has been involved in research and diagnosis of animal diseases since 1960.

Mebus' international experience includes teaching courses on animal diseases exotic to the Americas to veterinarians at FADDL and also in Argentina, Brazil, Cuba, Jamaica, and Venezuela. In addition, he has consulted on animal diseases in Cameroon, Egypt, Israel, and Niger. More than 100 of his research papers on animal diseases have been published in scientific journals, and he has authored numerous book chapters. Primary areas of research have been bovine neonatal diarrhea and animal diseases exotic to the United States.

While professor of veterinary pathology at the Universtiy of Nebraska, Mebus was the first to describe rotavirus and coronavirus as a cause of bovine neonatal diarrhea and to isolate the viruses.

Mebus will be missed by his VS colleagues. He will remain on Long Island to enjoy his retirement with his children and grandchildren and will pursue his favorite hobbies of fishing and gardening in his spare time.

## Foreign Animal Disease Update

This update consolidates into tables information from Office International des Epizooties (OIE) bulletins issued October 1994 through March 1995. Countries reporting disease outbreaks are listed below the appropriate disease heading (followed by the month/year of the report and total number of outbreaks reported for that time period). The notation "+" indicates that the presence of disease was reported without information on total number of outbreaks. Outbreak number followed by "+" indicates number of outbreaks as well as the presence of disease.

### Foot-and-Mouth Disease

*Virus Untyped*
Benin (1–3,7&10–12/94) 6
Bhutan (7–10&12/94–1/95) 8+
Bolivia (2,4–7,10&12/94) 18
Brazil (5–12/94) 1,222
Burkino Faso (7–12/94) 57
Cambodia (1–6/94) +
Chad (6–12/94) 14+
Eritrea (2&3/95) 4
Ethiopia (1–6/94) 12
Ghana (7–11/94) 20
India (7–10/94) 665*
Laos (4–9/94) +
Myanmar (9,10&12/94&1/95) 5
Nepal (1–9/94) +
Nigeria (9–12/94) +
Oman (9–12/94) 27+
Pakistan (11&12/94) +
Peru (1–12/94) 63
Tanzania (1–6/94) 29
Thailand (10–12/94&1/95) 6
Togo (5–9/94) 6
Venezuela (11/94) 1

* Elephant.

*Virus O*
Bolivia (1–4,6–9&11/94) 11+
Brazil (5–12/94) 159
Cambodia (9/94) +
Colombia (8/94–2/95) 168
Ecuador (8&10/94–2/95) 8
Ethiopia (5&8/94) +
Greece (10/94) 1
Hong Kong (11/94–1/95) 3+
Iran (5–10/94) 104
Israel (3/95) 1
Jordan (2/95) 1
Kenya (2/95) 1
Kuwait (1–5&8/94) 38
Malaysia (peninsula) (7–12/94&3/95) 8+
Pakistan (11&12/94) +
Paraguay (5&9/94) 4
Peru (1–9/94) 22+
Philippines (9/94–1/95) +
Saudi Arabia (10/94&2/95) +
Sri Lanka (1/95) 1
Thailand (9/94–1/95) 12
Tunisia (8/94) 1
Turkey (9/94–2/95) 25

*Virus A*
Bolivia (1&4/94) 2
Brazil (5–9&11–12/94) 53
Colombia (9/94–2/95) 45
Ethiopia (3/94) +
Kenya (1/95) 1
Pakistan (11&12/94) +
Philippines (8/94) +
Venezuela (5–9&11/94–1/95) 6

*Virus C*
Bolivia (5/94) 1
Brazil (5,6&8/94) 3
Philippines (9/94) +

*Virus SAT 2*
Uganda (6–9,11&12/94) 1+

*Virus SAT 3*
Namibia (10/94) 1

*Virus Asia 1*
Bhutan (11/94) +
Malaysia (peninsula) (9/94–2/95) 33+
Thailand (9/94–1/95) 15

### Hog Cholera

Belarus (2,5&10–12/94) 8
Belgium (10&11/94) 3
Brazil (5&7–11/94) 53
Bulgaria (9–11/94) 6
Cambodia (4–6/94) +
China (People's Republic) (7–12/94) 77
Croatia (10/94) 1
Cuba (9/94–1/95) 14+
Ecuador (1&2/95) +
FRY (Serbia & Mont.) (11/94–1/95) 12
FYR of Macedonia (10/94) 5
Germany (9/94–2/95) 41
India (7–10/94) 35
Italy (9–11/94&1–3/95) 26
Korea (Republic) (10/94–1/95) 9
Laos (4–9/94) +
Malaysia (peninsula) (8/94) 1
Mexico (11/94) 4
Moldavia (2&3/95) 3
Myanmar (11/94) 1
Paraguay (10&12/94) 2
Peru (1–8,10&11/94) 3+
Philippines (7/94–1/95) +
Russia (8–12/94) 13
Slovak Republic (9–11/94&1–2/95) 34
Taipei China (6,8&9/94&1/95) 6
Thailand (10/94–1/95) 4
Venezuela (3&4/94) 1
Vietnam (7–9/94) 37

### Lumpy Skin Disease

Benin (1–7&10–12/94) 6
Botswana (8–11/94) +
Burkino Faso (1–11/94) +
Comoros (7–10/94) +
Ethiopia (1/94) 1
Ghana (9/94) 3
Kenya (12/94–2/95) 2+
Mali (8/94) 4
Namibia (10&12/94–2/95) 5
Nigeria (9–12/94) +
Senegal (12/94–2/95) 3+
South Africa (10/94–3/95) 64
Swaziland (7/94–3/95) +
Tanzania (1–6/94) 83
Uganda (1–12/94) 12+
Zambia (3/94–3/95) +
Zimbabwe (10/94–3/95) 43

### Peste des Petits Ruminants

Benin (1–7&10–12/94) 15
Burkino Faso (1,3,4&6–12/94) 18
Cote d'Ivoire (12/94–1/95) 3
Eritrea (1–3/95) 12
Ethiopia (1/94) 1
Ghana (7–9&11/94) 12+
Guinea (10/94–2/95) +
India (9&10/94) 32
Israel (Controlled Territories) (10&11/94) 2
Nigeria (9–12/94) 13+
Oman (9–12/94) 9
Senegal (9/94–2/95) 5+
Togo (1–9/94) 33

### Vesicular Stomatitis

*Virus Not Typed*
Panama (10&11/94) 2
Venezuela (11/94–1/95) 6

*Virus Indiana*
Colombia (9/94–2/95) 63
El Salvador (8&9/94) 2
Panama (10/94) 1
Peru (10/94) 1

*Virus New Jersey*
Colombia (9/94–2/95) 127
El Salvador (8,9,11&12/94) 29
Honduras (8/94) 2
Mexico (10–12/94) 5
Peru (1–8&12/94) 2+
Venezuela (3–6&10–12/94) 8

### Contagious Bovine Pleuropneumonia

Benin (1–7&10–12/94) 12
Botswana (2/95) 5
Burkino Faso (1,7,10&12/94) 7
Cote d'Ivoire (12/94) 6
Ethiopia (1&3–5/94) 10
Guinea (10/94–2/95) +
Kenya (2/95) 1
Mali (2–7/94) 6
Namibia (11/94&1–3/95) 6
Nigeria (9–12/94) +
Tanzania (8/94) 3
Togo (7–9/94) 4
Uganda (1–12/94) 9+

**Newcastle Disease**
*Virus Not Characterized*
Albania (5/94–2/95) 5,059
Benin (1–6&10–12/94) 7
Brazil (5–7&10–12/94) 30
Burkino Faso (11/94) 1
Cambodia (4–6/94) +
Chad (6–12/94) +
China (People's Republic) (7–12/94) 70
Congo (9&10/94) +
Egypt (9/94&2/95) 3
Ethiopia (3/94) 1
FRY (Serbia & Mont.) (1/95) 1
Ghana (7–11/94) 26
Guinea (10/94–2/95) +
India (7–10/94) 87*
Iran (5–10/94) 134
Italy (10&12/94) 2
Jordan (9/94–2/95) +
Laos (4–9/94) +
Malaysia (peninsula) (7,8&10/94) 3
Myanmar (9,10&12/94&1/95) 4
Nepal (1–9/94) +
Nigeria (9–12/94) 1+
Pakistan (11&12/94) +
Peru (1–11/94) +
Philippines (7&11/94–1/95) +
Senegal (9–11/94&1–2/95) +
Sierra Leone (1–12/94) +
South Africa (10/94–3/95) +
Swaziland (12/94–3/95) +
Syria (5&7–12/94) 334
Tanzania (1–6/94) 111
Togo (1–9/94) 42
Uganda (1–3&5–12/94) +
United Arab Emirates (10/94) +
Uzbekistan (10/94) 1
Vietnam (7–9/94) +
Zambia (3/94–3/95) +

* Incomplete total.

*Velogenic Virus*
Botswana (8–12/94) +
Colombia (9/94) 2
Comoros (7–10/94) +
Germany (9/94–2/95) 55
Kenya (11/94&1/95) 6
Korea (11/94–1/95) 10
Malaysia (peninsula) (8,9&11/94) 4
Mauritius (4&5/94) 8
Namibia (2/95) 1
Netherlands (11/94) 1
Paraguay (8&11/94) 3
Russia (7&8/94) 3
South Africa (10/94–3/95) 85
Sri Lanka (9/94–2/95) 47
Sudan (3/95) 1
Taipei China (1–3/95) 30
Togo (6/94) 1
Tunisia (8&10–12/94) 26
Zimbabwe (11–12/94&2–3/95) +

**Sheep and Goat Pox**
Algeria (10/94–3/95) 544
Burkino Faso (6–9&12/94) 7
China (People's Republic) (7–12/94) 14
Eritrea (2&3/95) 6
Ethiopia (1–8/94) 34
India (7–10/94) 29
Iran (5–10/94) 107
Kyrgyzstan (9&10/94) 9
Morocco (10/94–3/95) 158
Nigeria (9–12/94) +
Oman (9&12/94) 9
Quatar (2/95) 47
Russia (12/94) 10
Senegal (1&2/95) +
Sri Lanka (12/94) 5
Syria (5&7–12/94) 195
Tunisia (8/94–1/95) 182
Turkey (9/94–2/95) 25
United Arab Emirates (10&11/94) 1+

**African Swine Fever**
Italy (10/94–3/95) 61
Kenya (10&11/94) 2
Mozambique (10/94) 3
South Africa (1/95) 1
Uganda (1–3&5–11/94) +
Zambia (10/94–3/95) +

**Fowl Plague**
Australia (12/94) 1
Benin (7/94) +
Comoros (7–10/94) +
Nepal (1–9/94) +

**African Horse Sickness**
Botswana (12/94) 1
Ethiopia (2/94) 2
Senegal (9,10&12/94–2/95) 4+

**Bluetongue**
India (8–10/94) 182
Israel (9–11/94) 12
South Africa (11/94&1–3/95) 28
United States (9/94–3/95) +
Zimbabwe (10/94&3/95) 2

**Rinderpest**
Eritrea (1/95) 1
India (7–10/94) 34
Kenya (2/95) 1
Uganda (5&7/94) 2

**Swine Vesicular Disease**
Italy (11/94–3/95) 16

(Dr. William White, International Services [IS], APHIS, USDA, 4700 River Road, Unit 65, Riverdale, MD 20737, 301–734–8892)

## Vesicular Stomatitis Virus–New Jersey (VSV–NJ) Epizootic in the Western United States—1995

**Disease Background.** Vesicular stomatitis is a viral disease that primarily affects cattle, horses, and swine during early spring, summer, and fall. The disease occasionally affects sheep, goats, and camelids. Many species of wild animals, including deer, bobcats, goats, raccoons, and monkeys, have been found to be experimentally susceptible hosts. Natural titers have been found in a wide range of free-ranging wild animals.

VSV is an RNA virus in the genus *Vesiculovirus*, family Rhabdovirdae. The virus is found in the Western Hemisphere. VS–NJ and VS–Indiana strains may be enzootic in the United States, Central America, Venezuela, Colombia, Ecuador, and Peru.

Vesicular stomatitis typically occurs in the United States in 10–15-year cycles in late spring through early fall. Summer climatic conditions throughout the Southwestern and Western United States appear to be optimal for incursions of vesicular stomatitis. VSV–NJ outbreaks have occurred in the United States in 1966, 1982–83, 1985, and 1995.

In affected livestock, vesicular stomatitis causes blisterlike lesions to form on the dental pad, tongue, lips, nostrils, coronary bands, and teats. These blisters swell and break, leaving raw tissue that is so painful that infected animals generally refuse to eat or drink and may show signs of lameness. Weight loss may follow, and, in dairy cows, a severe drop in milk production commonly occurs.

While vesicular stomatitis can cause economic losses to livestock producers, it is a particularly significant disease because its outward signs are identical to those of foot-and-mouth disease (FMD), which was eradicated from the United States in 1929. The clinical signs of vesicular stomatitis are also similar to those of swine vesicular disease. These diseases can be diagnosed and differentiated only through laboratory tests.

**Past Outbreaks in the United States.** During the 1982–83 outbreak, investigations were conducted on 1,324 premises, with 617 confirmed as laboratory positive (table 1). The disease appeared to follow the Rio Grande and Colorado River valleys through New Mexico and into western Colorado. By September 30, 1982, the disease had moved northward through Arizona, New Mexico, Colorado, Utah, Wyoming, Idaho, Montana, Nebraska, and South Dakota. Dispersal of dairy cattle in October and November of 1982 from Colorado and dispersal of cattle from Idaho apparently spread VSV–NJ to California, Washington, Oregon, Kansas, and Missouri.

**Table 1—Investigations conducted for vesicular stomatitis during the 1982–83 U.S. outbreak in the eight most affected States**

| State | Investigations Conducted | Positive Cases Found |
|---|---|---|
| Colorado | 453 | 313 |
| Idaho | 234 | 120 |
| Wyoming | 105 | 54 |
| New Mexico | 50 | 37 |
| Utah | 35 | 24 |
| California | 83 | 17 |
| Arizona | 25 | 11 |
| Texas | 60 | 0 |
| TOTAL* | 1,045 | 576 |

**Table 2—Investigations conducted for vesicular stomatitis during the 1985 U.S. outbreak**

| State | Investigations Conducted | Positive Virus Isolations | Positive Serology | Total Positive Cases |
|---|---|---|---|---|
| Colorado | 104 bovine | 32 | 23 | 55 |
| | 4 ovine | 0 | 0 | 0 |
| | 2 caprine | 0 | 1 | 1 |
| | 157 equine | 20 | 71 | 91 |
| | | | | 147 |
| Arizona | 2 bovine | 1 | 0 | 1 |
| | 1 ovine | 0 | 0 | 0 |
| | 4 equine | 0 | 2 | 2 |
| | | | | 3 |
| New Mexico | 38 bovine | 11 | 17 | 28 |
| | 51 equine | 3 | 33 | 36 |
| | | | | 64 |

The 1985 VSV–NJ outbreak was similar to the 1982–83 epizootic but with significantly smaller numbers of positive cases and a more narrow geographic distribution (table 2). The first case during this outbreak was confirmed on June 6, 1985, in New Mexico. Many of the 147 positive cases in Colorado were in the Pueblo area. The major focus of the 64 cases in New Mexico was in the Albuquerque region.

**Current Situation.** The index case of the 1995 VSV–NJ outbreak, confirmed by NVSL on May 6, was in an equine located in Las Cruces, NM. The current outbreak, which has affected New Mexico, Arizona, Colorado, Texas, Utah, and Wyoming, appears similar to the 1985 outbreak with regard to geographic distribution. The 1995 epizootic is more geographically confined than the 1982–83 epizootic but similar in location to the 1985 epizootic (see figs. 1–3).

A Livestock Disease Investigation Unit was established in Las Cruces on June 1, 1995. As the disease moved north/northwest, consistent with its historical movement, the unit was relocated to Englewood, CO, on July 2, 1995, and renamed the Livestock Disease Reporting Unit (LDRU). New Mexico State activities were still coordinated through the VS area office in New Mexico.

As of October 25, 1995, 996 premises had been investigated for vesicular stomatitis. Also as of that date, there had been 340 case-positive premises identified: 186 in New Mexico (181 had been released from quarantine), 139 in Colorado (95 had been released from quarantine), 1 in Arizona (released

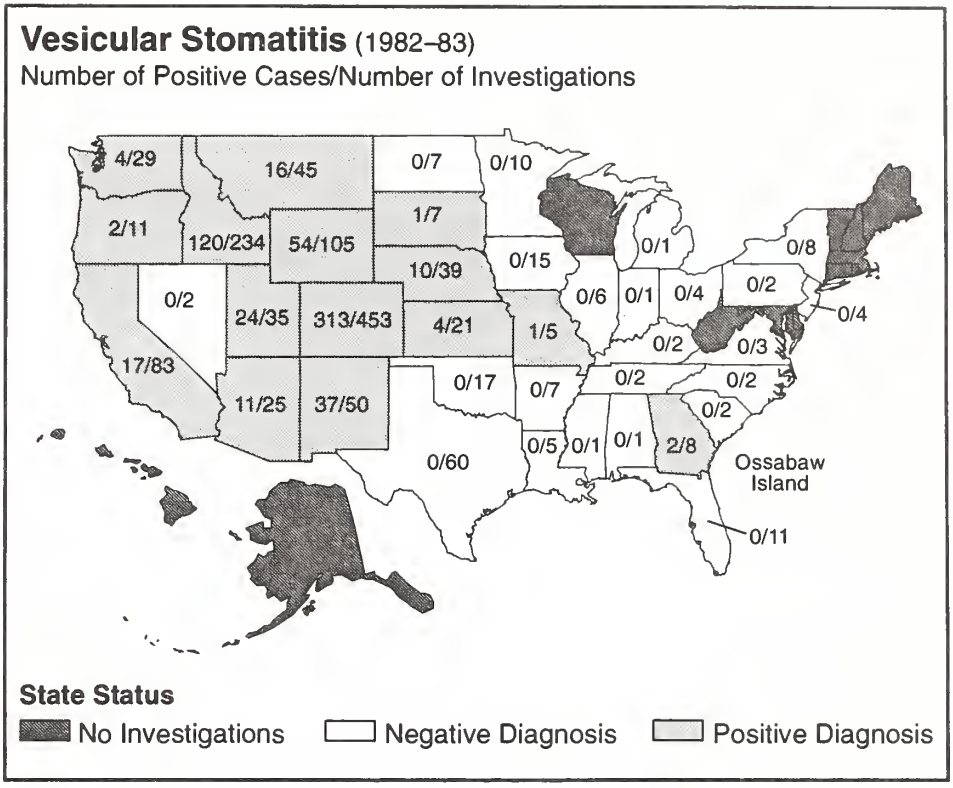


**Figure 1**—Data from the 1982–83 vesicular stomatitis outbreak in the United States. The first number in each State represents the number of positive cases found; the second number indicates the number of investigations conducted.

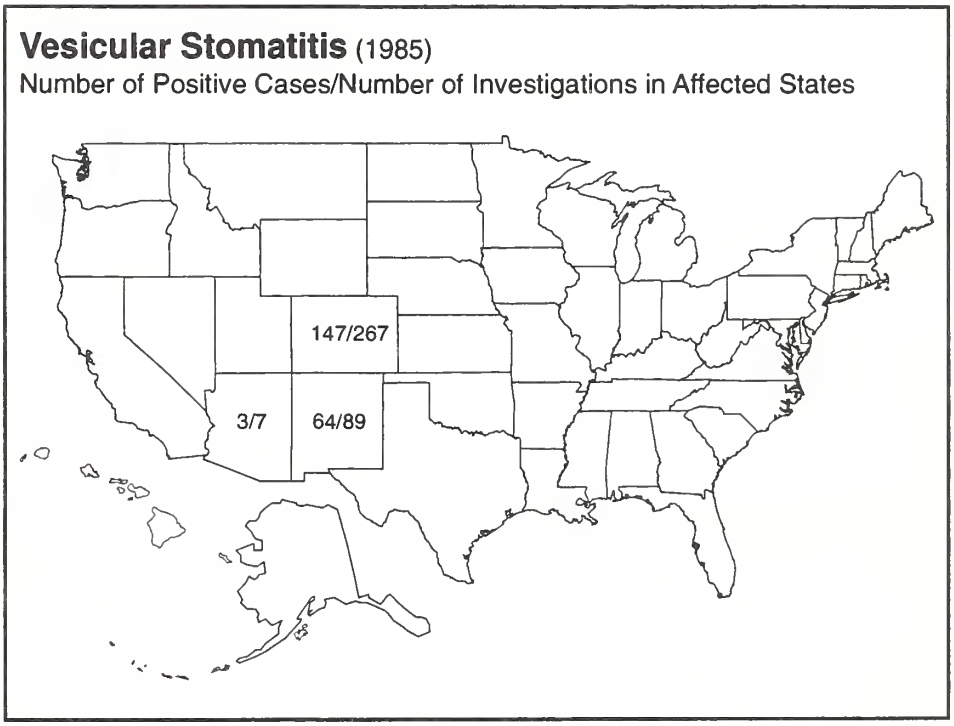


**Figure 2**—Data from the 1985 vesicular stomatitis outbreak in the United States. The first number in a State represents the number of positive cases found; the second number indicates the number of investigations conducted.

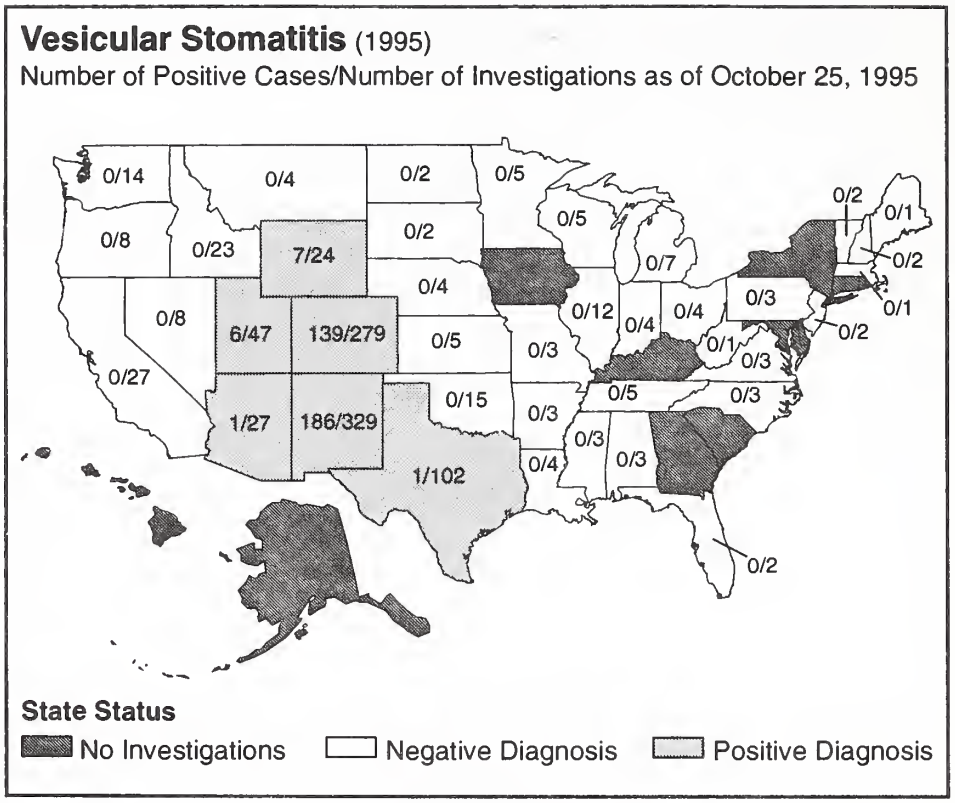


**Figure 3**—Data from the 1995 vesicular stomatitis outbreak in the United States. The first number in a State represents the number of positive cases found; the second number indicates the number of investigations conducted as of October 25, 1995.

from quarantine on August 7), 1 in Texas (released from quarantine on August 28) (Note: VSV–NJ was isolated from a second premises in Texas October 2, but because NVSL could not reisolate the virus, a second quarantine release date of October 16 is recorded for Texas without a second case.), 6 in Utah (2 released), and 7 in Wyoming. (See table 3 and fig. 4.) The major focus of the disease in New Mexico was in the Albuquerque area; in Colorado, it was in the Grand Junction area.

**Table 3—Statistics of the 1995 U.S. outbreak of vesicular stomatitis as of October 25**

| State | Current Positive Premises | Cumulative Positive Premises |
|---|---|---|
| Arizona | 0 | 1 |
| Colorado | 44 | 139 |
| New Mexico | 5 | 186 |
| Texas | 0 | 1 |
| Utah | 4 | 6 |
| Wyoming | 7 | 7 |
| Total | 56 | 340 |

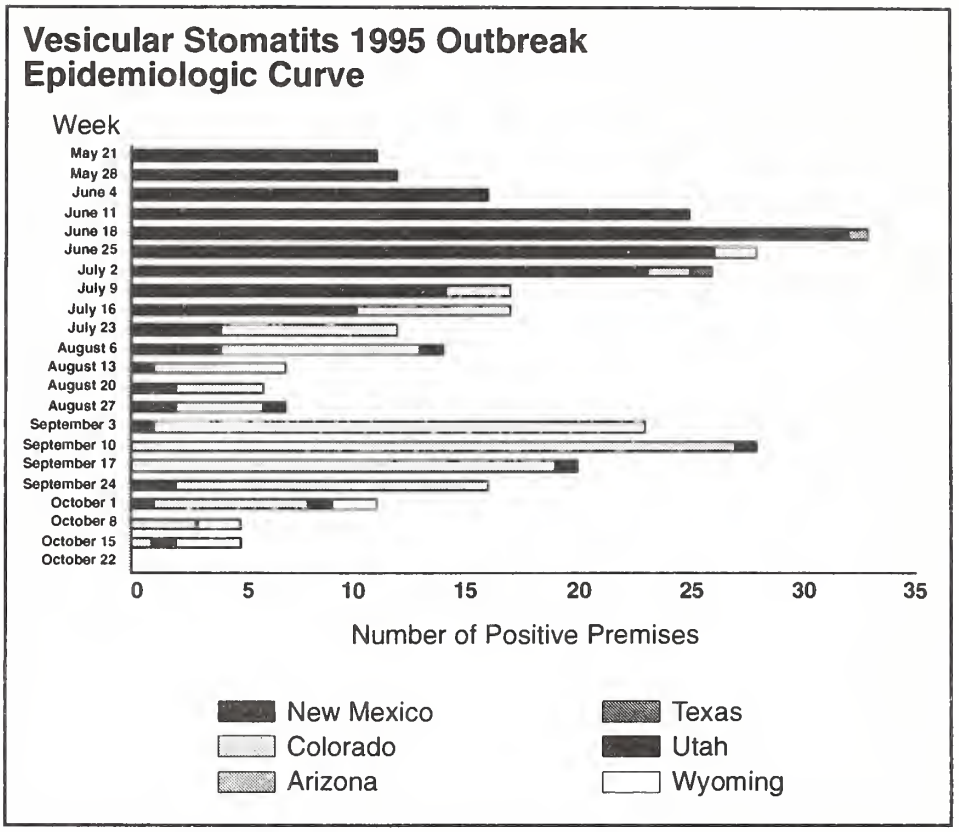


**Figure 4**—Epidemiologic curve of the 1995 vesicular stomatitis outbreak in the United States.

**Epidemiology**. Various theories and hypotheses regarding the epidemiology and spread of VSV–NJ have been articulated. Strong scientific data suggest that arthropod vectors may transmit the disease by both biological and mechanical means. Evidence also clearly supports animal movement and contact as ways to transmit the disease. An isolated theory relevant to a plant virus has also been postulated. This theory suggests that a plant virus modified by insects is transmitted to cattle and horses by these insects feeding on animals.

The one equine case in Texas in 1995 was unequivocally attributed to animal movement from an affected State. Significant movement of the disease to several States in 1982–83 was also attributed to movement of animals. Vectors, animal movement, and contact all may be important in the spread of the disease. Studies currently under way should provide information relevant to the spread and epidemiology of the 1995 VS outbreak (see Research and Epidemiologic Studies, below).

During the 1995 outbreak, primarily horses and backyard cattle have been affected. One llama was clinically affected with small oral erosions, positive serological results, and positive virus (VSV–NJ) isolation. The majority of lesions in equines have been on the lips and muzzle, where edematous swelling and scabs have occurred. Other lesions have included intact and ruptured vesicles, erosions, and ulcers present on the tongue and gums. One mare had ulcers, erosions, and scabs on the teats, and the nursing foal had oral lesions. One donkey had coronary band lesions, and one seropositive horse had lesions on the prepuce. The majority of cattle have had oral lesions

similar to those described for equines. Several cows have had teat lesions as well as interdigital hoof lesions.

Clinical specimens collected from the field have included serum, vesicular fluid, epithelial tags, and oral swabs. Equine specimens have been sent to NVSL, Ames, IA, for testing, while ruminant species have been forwarded to NVSL–FADDL, Plum Island, NY. Routine NVSL laboratory testing for VSV–NJ and VSV–Indiana has included complement fixation (CF) and serum neutralization (SN) testing of serum and virus isolation on vesicular fluid, epithelial tags, and oral swabs. Specimen testing at FADDL has included bluetongue agar gel immunodiffusion, FMD virus infection associated antigen, CF and SN on serum, and virus isolation on swabs and vesicular fluid.

From June 15, 1995, through October 25, 1995, cases, premises, and laboratory results were classified as follows:

(1) Investigation: The inspection of a premises with an animal or animals reported with clinical signs or lesions considered suspicious or compatible with VSV.

(2) Negative: An investigation in which the NVSL or FADDL laboratory results reported no response to VSV.

(3) Positive: An investigation in which the NVSL or FADDL laboratory results reported the isolation of VSV or a positive CF test in an animal demonstrating compatible clinical signs.

After vaccination was initiated in some States, this terminology was modified on August 21, 1995, as follows:

Classification criteria—unvaccinated animals:

(1) Negative: Serology results are CF negative, and virus isolation (if submitted) is negative at final reading. If these tests are negative and virus neutralization is positive, the case may be classified as negative or as a pending investigation.

(2) Positive: Requires either isolation of the virus or a positive field diagnosis and a positive CF titer (>5).

(3) Pending investigation: Serology and virus isolation tests are not complete. This status is not converted to positive or negative until all pending tests are completed. If the field diagnosis and laboratory results do not allow a positive or negative status classification (field diagnosis negative, serology positive, or vice versa), additional evaluation of the premises and additional serology and/or virus isolation attempts may be necessary. A positive virus neutralization, with negative CF and/or virus isolation, suggests the necessity for additional investigation.

Classification criteria—vaccinated animals:

(1) Negative: Serology is CF negative, and virus isolation (if submitted) is negative at final reading.

(2) Positive: Requires either isolation of the virus or field diagnosis and premises evaluation that support vesicular stomatitis infection and positive CF results.

(3) Pending investigation: Field evaluation, serology, and virus isolation tests are not complete. This status is not converted to positive or negative until all pending tests or field evaluations are completed.

In the absence of virus isolation, the field diagnosis and premises evaluation are the primary factors used in classifying the case status. A CF titer is not assumed to be a vaccine-produced titer unless the field diagnosis and premises evaluation support the absence of vesicular stomatitis.

**Quarantines and Restricted Circles and Zones.** Officials of affected States have placed affected premises under quarantine and also have instituted various animal movement restrictions. Quarantines remain in place until 30 days after healing of the last clinical lesion. In addition, some State officials have placed a 10–20-mile restricted circle around the quarantined premises.

A restricted circle is an area with a 10-mile radius around a quarantined premises. Within a restricted circle or zone, prohibited activities include movement of animals, shipments of livestock to other States, and gatherings of animals (auction markets, rodeos, fairs, etc.).

Early in the outbreak, due to the abundance of positive premises in and to the south of Albuquerque, an approximate 2,000-square-mile restricted zone was established to prevent disease spread. This zone extended 10 miles on either side of the Rio Grande River and from State Highway 44 on the north to U.S. Highway 380 in the south. This zone was lifted on July 26, 1995.

Most States have implemented temporary restrictions regarding importation of livestock from States affected by VSV. The basic and most common requirement is a statement on the health certificate similar to the following: "Vesicular stomatitis has not been diagnosed within 10 miles of the premises of origin within the last 30 days. I have examined the animal(s) and have found no signs of vesicular stomatitis." Other restrictions implemented by some States include requirements for permits for entry, quarantines postentry, negative VSV tests, total bans on livestock from affected States, and prohibition of VSV-vaccinated animals.

**International Restrictions.** Some international trade restrictions were implemented in response to the diagnosis of VSV–NJ in the United States. These restrictions, as of October 24, 1995, are as follows:

(1) Canada: Certification that horses, ruminants, and swine have not been in a VSV-affected State (Colorado, New Mexico, Utah, or Wyoming) for the past 30 days. All horses entering must be inspected by a Canadian Federal veterinarian. Livestock from Arizona and Texas must have a negative CF VSV test and have been resident on an unaffected premises with protection from insect vectors for 30 days prior to export.

(2) European Union: Certification that live horses have a negative SN VSV test with a statement that, for the past 30 days, they have not been in any State or had contact with any livestock that has been in any State affected with VSV within 6 months.

(3) Russia: No U.S. beef from affected States.

(4) Romania: No U.S. beef.

(5) South Africa: No U.S. beef or pork from affected States.

(6) Chile: Live susceptible animals must be from a VSV-free State.

(7) United Arab Emirates: Same as European Union.

Although there are many similarities between the 1995 VSV–NJ outbreak and previous epidemics, one difference in 1995 is in the area of trade relations. The General Agreement on Tariffs and Trade, the North American Free Trade Agreement, and the European Union are now fully organized and operational. The VSV–NJ diagnostic, surveillance, and control operations were designed to conform to this new world trading order.

**Vaccine**. An autogenous, killed VSV–NJ equine and bovine vaccine prepared by Grand Laboratories, Larchwood, IA, was released to the affected States of New Mexico, Arizona, and Colorado on July 18, 1995. Other bordering States have requested use of the vaccine. USDA–APHIS–VS approved additional vaccine use in Utah and Wyoming on August 9, 1995. Vaccine use in Idaho was approved on August 15, 1995. As of September 1995, approximately 6,400 equine doses and 18,000 bovine doses of vaccine have been sold. Each State is responsible for maintaining current, accurate vaccination records in their respective State.

Each vaccine shipment is accompanied by a detailed, extensive factsheet that includes information relevant to production and use. Specific requirements have been defined by USDA for States using the vaccine. These requirements include

(1) Adverse reactions must be reported immediately to the USDA–APHIS–Veterinary Biologics Field Office hotline. (None have been reported.)

(2) Vaccination records must be kept on all animals vaccinated. Individual animal identification is required.

(3) Vaccination is recommended for dairy herds at high risk.

(4) A VSV outbreak in a vaccinated herd should be reported immediately.

(5) Vaccination does not supersede existing State regulations relevant to animal movement and quarantine.

**Research and Epidemiologic Studies.** Team-oriented VSV–NJ research is being planned and conducted. This information should be useful for future

outbreaks. Research efforts are being undertaken by ARS; VS' Centers for Epidemiology and Animal Health, NVSL, and NVSL–FADDL; as well as the University of Georgia, Southeastern Cooperative Wildlife Disease Study (Athens, GA).

Epidemiologic studies are planned: (1) detailed epidemiologic case investigations, (2) a case-control study, (3) entomological studies, (4) wildlife seroprevalence, (5) geographic information systems (GIS) studies, (6) a vaccine field study regarding seroconversion, and (7) molecular epidemiology.

(1) Detailed Epidemiologic Case Investigations. The objectives of this study are to develop a detailed picture of each infected premises and intraherd epidemiology. Potential risk factors in disease spread may be identified.

Methods: Serial serum samples will be collected from each animal in the herd for antibody titer analysis and calculation of seroconversion rates. Clinical signs exhibited by all infected animals will be assessed and documented. Insects and rodents will be collected for virus isolation. Plant types on pastures will be evaluated along with local geographical features. Priority will be assigned to dairy farms as well as premises infected in previous outbreaks. Numbers of personnel and investigations will depend on availability of resources.

(2) Case-Control Study. The objective is to evaluate management factors to identify risk factors that may predispose herds to infection.

Methods: Followup investigation of premises will be conducted via telephone interviews. All bovine investigations and index equine investigations in New Mexico, Colorado, and Utah will be included. Investigations that were not classified as cases because the animals were not considered to be infected will serve as controls. Priority will be given to the most recent investigations followed by those in chronological order as far back as possible.

(3) Entomological Studies by ARS. These studies will determine the role of vectors in maintaining virus presence during epizootic and nonepizootic periods.

Methods: Projected as long-term (3–5 years) studies, ARS will revisit infected premises in years subsequent to the outbreak. The study will include infected premises with no evidence of exposure to other potentially infected animals (based on field data and results of the telephone survey), repeat cases from previous outbreaks, and geographically isolated premises.

(4) Wildlife Seroprevalence. These studies will assess the presence in wildlife species of antibodies that would indicate exposure to VSV. Such exposed species may then serve as enzootic or epizootic reservoirs of the virus.

Methods: Wild rodents and game animals present in the area of selected herds could be serologically tested for evidence of VSV infection. Criteria for inclusion will be the same as in the entomological studies.

(5) GIS Studies. These studies will identify geographical features that put premises at risk of VSV infection.

Methods: Data on latitude and longitude will be merged with available data on vegetation, waterways, altitude, etc., and analyzed for common features found in case premises.

(6) Vaccine Field Study. The objective is to determine the seroconversion rate of vaccinated animals.

Methods: Change in seroprevalence rates will be measured before and after single and double doses of autogenous vaccine. Number of herds to be tested will depend on the choice of the owners.

(7) Molecular Epidemiology. The objective is to identify the original source of the 1995 epizootic.

Methods: Bovine and equine isolates from the 1995 epizootic will be compared to the 1995 isolate from Hidalgo, Mexico, the 1985 U.S. outbreak, and the 1982–83 U.S. outbreak. Isolates from each State in the 1995 outbreak may be used. Laboratory work could begin after cases during the current outbreak have declined or as time becomes available.

**Reporting Communications.** The LDRU continues to produce a daily tabulation of new cases, quarantines released, cases closed, and other demographic data. USDA–APHIS–IS continues to report relevant data on VSV–NJ to OIE. APHIS maintains a toll-free telephone number (1–800–545–USDA) for the public and industry to use to obtain daily VSV updates. During a 1-week period in August 1995, there were 1,205 calls to this number. APHIS–Legislative and Public Affairs has issued numerous news releases on VSV. Various informative materials have also been widely distributed, including factsheets on vesicular stomatitis; dairy, beef, equine, and swine vesicular stomatitis; and vesicular stomatitis vaccine and a vesicular stomatitis vs. FMD color pamphlet.

For the first 3 months of the outbreak, a daily conference call was held among APHIS staffs. Many conference calls have also been held between APHIS and industry, State, and Federal officials to discuss issues such as vaccination, research, quarantines, and restricted circles.

A conference call was being held weekly with industry livestock and health officials, the LDRU, and VS–Emergency Programs, VS–National Center for Import and Export, IS, and other relevant staff. Industry groups that have participated include the National Milk Producers Association, National Cattlemens Association, American Veterinary Medical Association, Association of American Veterinary Colleges, American Farm Bureau Federation, Government Industry Affairs Livestock Marketing Association, National Horse Council, National Pork Producers, and other groups. A weekly conference call is still being held between APHIS personnel and the affected Western State Veterinarians.

(Compiled by Dr. Terrance Wilson, Emergency Programs, VS, APHIS, USDA, 4700 River Road, Unit 41, Riverdale, MD 20737, 301–734–8073)

## Bovine Spongiform Encephalopathy (BSE) Update

**Domestic Surveillance**. Surveillance for BSE in the United States continues. An additional 133 brains were received by NVSL for examination from April 1 to June 30, 1995, bringing the total number of brains that have been submitted for examination to 2,291 as of June 30, 1995 (fig. 5). No histopathological evidence of BSE has been found in any U.S. cattle.

**U.K. Update.** Great Britain reported 7,569 newly confirmed cases of BSE with 543 more herds affected between March 3 and September 1, 1995 (table 4). Review of the epidemic curve (fig. 6) indicates that the epidemic continues to decline.

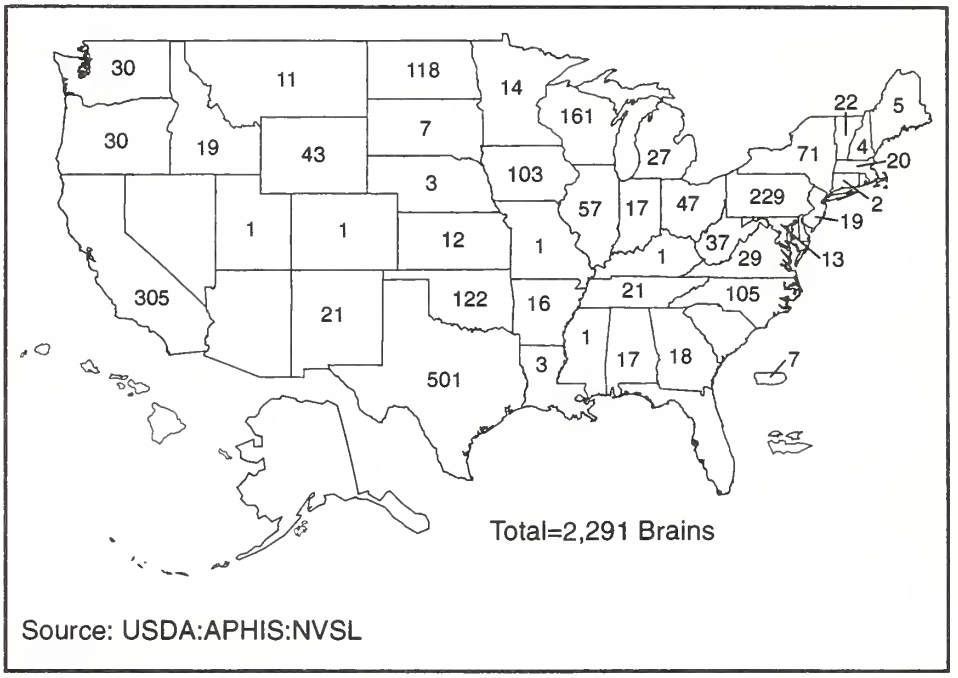


**Figure 5**—Total U.S. bovine brain submissions for BSE testing by State, 1986—June 30, 1995. (Note: None of these submissions have tested positive for BSE.)

**Table 4—Descriptive epidemiologic statistics for BSE in Great Britain* as of September 1, 1995**

| | |
|---|---|
| Total number of confirmed cases | 152,470 |
| Total number of affected herds | 32,719 |
| Percentage of dairy herds affected | 53.8 |
| Percentage of beef suckler herds affected | 15.0 |

* England, Scotland, and Wales.
Data provided by Great Britain.

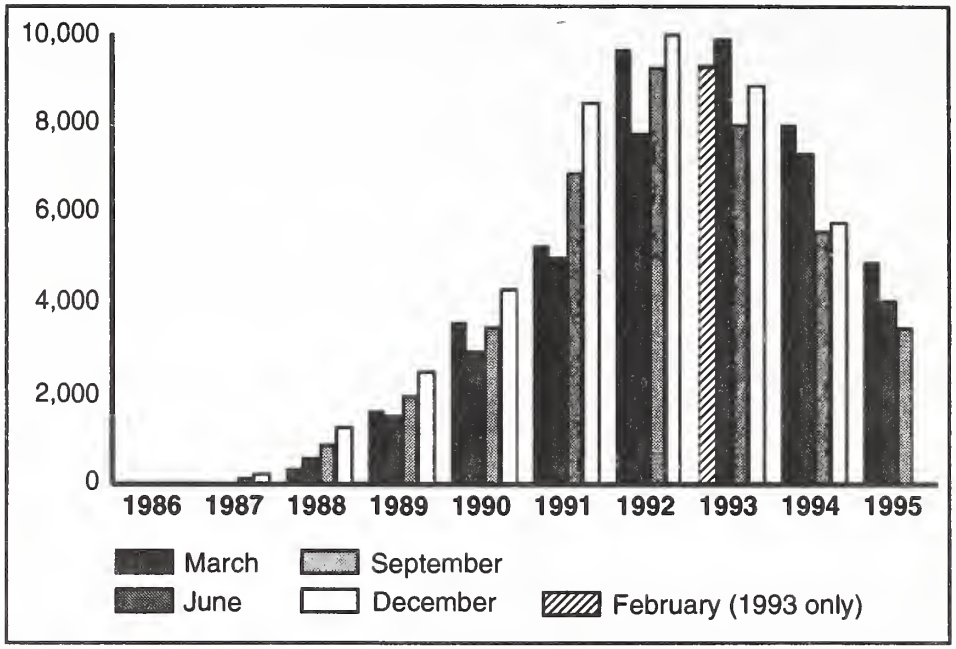


**Figure 6**—Number of new cases of BSE in Great Britain, September 1986—September 1995.

**Table 5—BSE cases[1] worldwide other than Great Britain as of August 9, 1995**

| Country[2] | 1987 + before | 1988 | 1989 | 1990 | 1991 | 1992 | 1993 | 1994 | 1995 | Total |
|---|---|---|---|---|---|---|---|---|---|---|
| Guernsey | 4 | 34 | 52 | 83 | 75 | 92 | 115 | 69 | — | 524 |
| Northern Ireland | 0 | 3 | 30 | 100 | 170 | 333 | 487 | 363 | 112[3] | 1598 |
| Jersey | 0 | 1 | 4 | 8 | 14 | 23 | 37 | 22 | — | 109 |
| Isle of Man | 0 | 6 | 6 | 22 | 67 | 109 | 110 | 55 | — | 375 |
| Republic of Ireland | 0 | 0 | 15 | 14 | 17 | 18 | 16 | 19 | 3[3] | 102 |
| Switzerland | 0 | 0 | 0 | 2 | 8 | 15 | 29 | 64 | 52[3] | 170 |
| Portugal | 0 | 0 | 0 | 1[4] | 1[4] | 1[4] | 3[4] | 12 | 10[3] | 28 |
| France | 0 | 0 | 0 | 0 | 5 | 0 | 1 | 4 | 2 | 12 |

Countries with imported cases only:
Germany: 4 cases (2/92, 2/94, 4/94, 5/94)
Falkland Islands: 1 case (1989)
Canada: 1 case (11/93)
Italy: 2 cases (10/94)
Denmark: 1 case (7/92)
Oman: 2 cases (1989)

1. Cases in native cattle and cattle imported from the United Kingdom or another country with endemic BSE.
2. In order of first reported case/diagnosis.
3. Data for Northern Ireland as of September 1, 1995; data for Switzerland as of August 25, 1995; data for Portugal as of August 1, 1995; data for the Republic of Ireland as of March 31, 1995.
4. Imported cases.

Data provided by OIE and Northern Ireland.

**Other BSE-Affected Countries.** Between April 8 and August 1, 1995, Portugal reported nine additional cases of BSE in native cattle. France reported one additional native case between April 8 and June 12, 1995. Switzerland reported 33 additional cases in native cattle between March 25 and August 25, 1995. Northern Ireland reported an additional 69 cases in native cattle between March 6 and September 1, 1995, and the Republic of Ireland reported 3 additional cases of BSE in native cattle between March 31 and June 12, 1995 (table 5). No additional reports of cases of BSE imported from the United Kingdom or other countries with endemic BSE were recorded since the last reporting period.

(Source: DxMonitor, Summer 1995, USDA, APHIS, VS, Centers for Epidemiolgy and Animal Health, 555 South Howes, Fort Collins, CO 80525 970–490–8000)

## Highly Pathogenic Avian Influenza (HPAI) Virus in Mexico—Update

As of September 5, 1995, the HPAI outbreak in Mexico involved 17 poultry farms that are quarantined in the affected States of Queretaro and Puebla. Approximately 40 additional flocks are quarantined in 9 additional States and are assumed to be affected with low pathogenic or medium pathogenic avian influenza.

Mexican animal health officials continue their national eradication campaign. Extensive vaccine use continues in the States of Queretaro, Puebla, Jalisco, and Guanajuato. Limited vaccine use is being reported in the States of Hidalgo, Michoacan, and Veracruz.

The Mexican States that border the United States are voluntarily testing and depopulating seropositive flocks. One poultry company in the Mexican State of Nuevo Leon depopulated, cleaned and disinfected, and final-tested a cluster of 16 broiler farms.

A survey for avian influenza conducted by Mexican animal health officials in July 1995 involved the six Mexican States of Nuevo Leon, Tamaulipas, Coahuila, Jalisco, Nayarit, and Zacatecas. This survey was expanded in August 1995 to add additional Mexican States. Survey findings resulted in the quarantine of approximately 60 farms in 11 Mexican States.

(Dr. Kelly Preston, Emergency Programs, VS, APHIS, USDA, 4700 River Road, Unit 41, Riverdale, MD 20737, 301–734–8073)

## Taura Syndrome of Farmed Penaeid Shrimp

**Introduction.** Over the past few years, Taura syndrome (TS) has emerged as an important factor affecting the economic viability of shrimp farming in the Americas. Estimates of the annual impact of TS to shrimp farms in Ecuador range over $100 million. First reported in 1992 on shrimp farms near the Taura River, Gulf of Guayaquil, Ecuador, TS is currently recognized in many shrimp farming regions in the Americas.

**Etiology.** Initially the cause for TS was attributed to exposure to the fungicides Tilt (propiconazole, Ciba–Geigy) and Calixin (tridemorph, BASF). Although the role of fungicides in the etiology of TS now seems remote, researchers in Ecuador continue to maintain that fungicide exposure causes TS.

More recently, researchers have identified a virus in shrimp affected by TS. The TS virus (TSV) has been shown to be the direct etiologic agent of the disease. Virions of TSV have an icosahedral morphology, a diameter of 31–32 nm, and a buoyant density of about 1.337 g/ml. These features, and a nucleic acid that is a linear single-stranded RNA, are consistent with placement of TSV in the family Picornaviridae. Studies on TSV recovered from cultured shrimp epidemics in Ecuador and Hawaii have shown the same virus to be present in both locations.

**Clinical Attributes and Pathology.** Typically, TS occurs in juvenile (0.1 to 5 g) Pacific white shrimp *(Penaeus vannamei)* within 2 to 4 weeks of stocking into nursery or growout ponds or tanks. TS is also known to occur in tank-held postlarvae as well as subadult to broodstock *P. vannamei.* The clinical effect of TS is mortality. Within shrimp populations in ponds on a particular farm, the course of TS may be acute (5 to 20 days) to chronic (over the period of growout, e.g., 120 days), and the cumulative mortality variable is 5 to nearly 100 percent. Reasons behind the variability in attack rate in shrimp ponds are largely undefined.

Shrimp species and age are recognized as susceptibility factors. *P. vannamei* is sensitive to TS, while the Pacific blue shrimp, *P. stylirostris,* is refractory. In general, TSV challenge of juvenile *P. vannamei* results in more severe disease expression than if shrimp initially contract TSV as older animals. For example, while peak mortality of greater than 90 percent is not unusual for TS-affected juvenile *P. vannamei,* cumulative mortality generally does not exceed 50 percent for adult *P. vannamei.*

During an outbreak, dead and dying shrimp will appear in seines or cast nets used for routine population sampling. In the case of high-density, clearwater raceways in Hawaii, affected shrimp were visible on the bottom or in the water column of the shallow culture tanks. Predatory birds are often attracted to TS-affected ponds and feed heavily on moribund shrimp. On farms where pond populations are not periodically sampled, some outbreaks may go unrecognized until a pond is harvested. These cases present with low survival and unexplained disappearance of shrimp. Surviving shrimp from a TSV epidemic are usually outwardly and histologically free of signs and lesions of TS.

For individual shrimp, the course of TS appears to occur largely within the period of a single molt cycle. Juvenile *P. vannamei* in the peracute to acute phase of TS are weak, have a soft shell and an empty digestive tract, and usually have diffuse expansion of red chromatophores. These shrimp typically are in late premolt to early postmolt stage. In clearwater raceways in Hawaii, TS-affected shrimp, when viewed from above, had a slightly opaque appearance to the cuticle, and the red coloration was not usually pronounced, although chromatophore expansion was present. Shrimp in the peracute/acute phase of TS have multiple foci of epidermal and subepidermal necrosis, the histopathology lesion diagnostic for TS. However, acute TS lesions are not usually readily apparent to the naked eye.

Individuals that survive an acute episode of TS and progress into the intermolt stage of the molt cycle have multiple, randomly distributed, irregularly shaped, pitted, melanized lesions of the cuticle. Apparently, these foci are sites of regeneration and healing of the acute lesions. These symptoms designate the chronic phase, the start of recovery from the infection. Shrimp in the chronic phase usually display normal behavior.

Shrimp in the chronic phase that pass through premolt and ecdysis shed the outer cuticle with the melanized foci. Typically, these shrimp will not have melanized cuticle lesions, but some animals may have pale, depigmented foci in the cuticle epidermis. The pale foci probably represent previous sites of cuticle necrosis, and chromatophore regeneration has been delayed. These shrimp are considered to be in the recovery phase of TS and lack either the gross or the microscopic lesions diagnostic for TS. Bioassay test findings on asymptomatic subadult *P. vannamei* have indicated TS recovery phase shrimp can be chronic carriers of TSV. Moreover, it is felt that *P. vannamei* in the recovery phase are less susceptible to recurrence of TS.

The histopathology lesions associated with TS in shrimp are described in detail. The histological changes that characterize TS in the acute form are multifocal areas of necrosis in the cuticular epithelium and, often, the subcuticular connective tissue and, at times, the underlying striated muscle. Nuclear pyknosis and karyorrhexis are common, as are multiple prominent, spherical inclusion bodies that range from 1 to 20µm in diameter. These cytoplasmic inclusions are scattered throughout the affected areas of cuticle tissue and impart a "buckshot" appearance, stain eosinophilic to basophilic in hematoxylin and eosin preparations, and are Feulgen negative. Occasionally, necrosis of antennal gland epithelium is observed. *P. vannamei* sampled in the acute phase of TS from across the geographic range and environmental conditions where this disease has occurred have had identical histopathological lesions of the integument. Histopathologic lesions indicative of other chronic or subclinical shrimp diseases are commonly encountered in shrimp examined from TS epidemics.

Ultrastructural features for focal, peracute TS lesions include spherical intracytoplasmic inclusions with contents that ranged from electron lucent to electron dense, which correspond to the buckshot appearance of the cytoplasm of such cells as seen in histological preparations, and, in some preparations in which a calcium phosphate buffer is used for fixation, the presence of needlelike crystals. Polyribosomes and partially formed virus particles about the size of ribosomes are generally abundant in these lesions. Fully formed virions can be difficult to see in electron microscopy preparations of acute TS lesions.

**Diagnosis.** At present, the diagnosis of TS is made by histopathologic demonstration of the buckshot pattern of necrosis in the epidermal and subepidermal tissue of the integument of the gills, appendages, or general body cuticle. The pathogonomic histopathology changes are usually most reliably present in shrimp in the peracute/acute phase of TS. Shrimp in the chronic phase may or may not have acute phase lesions. Chronic phase TS lesions share gross and histopathologic features with bacterial shell disease (e.g., black spot) and may be difficult to differentiate solely on gross or microscopic criteria. Shrimp in the recovery phase or asymptomatic animals from ponds with active TS will lack

the microscopic pathological changes necessary for a dependable diagnosis. Therefore, submission of specimens to a diagnostic laboratory should include a nonrandom selection of shrimp in the peracute to acute phase of TS. These specimens should be properly preserved and killed by injection of Davidson fixative or be stored and submitted frozen for evaluation by indicator shrimp TSV bioassay.

On farms with a history of TS, the highly suggestive TS gross pathological changes typical for the peracute/acute and chronic TS are sufficient to make a field diagnosis. However, gross pathological changes alone are not reliable as a sole means of diagnosis for occurrences of the disease in new locations.

Indicator shrimp bioassay (ISB) is another approach for the diagnosis of TSV. This procedure affords an opportunity to detect TSV from shrimp populations where peracute and acute phase specimens are infrequent or to evaluate nonshrimp substrates. The bioassay procedure relies upon transmission of viable TSV to the highly susceptible, specific-pathogen-free Mexican strain *P. vannamei*. Indicator shrimp are either fed or injected with a filtered extract of the test substrate. The incubation period varies from a few days (injection exposure) to 5 to 10 days (oral exposure). Once infected, some of the indicator shrimp undergo the peracute/acute phase of TS and can be collected, preserved, and sectioned to demonstrate foci of necrosis in the cuticle epidermis. Although the sensitivity of ISB has not been established, the procedure has been found clearly to indicate the presence of virulent TSV from shrimp sampled from chronic and recovery phase stages of TSV infection.

Enhancement of shrimp is another option for demonstration of TSV. Enhancement has been applied for assessment of capture wild broodstock. In this case, shrimp of unknown TSV status are held crowded for 10 to 20 days and observed daily for signs of TS. A rise in daily mortality, the presence of the diagnostic histopathology changes in the cuticle epidermis, and shrimp with chronic phase lesions are used as criteria for a diagnosis of TSV.

Recent efforts in the laboratory of Dr. Don Lightner, University of Arizona, have yielded a gene probe for TSV. Currently, this probe is undergoing field testing and refinement.

**Geographic Range.** Shrimp Farms. TS was first recognized in June 1992 in farms near the mouth of the Taura River in the Gulf of Guayaquil, Ecuador. The disease became widespread on farms in the Gulf and was identified in Tumbes, Peru, by midsummer 1993. TS was also identified in *P. vannamei* farmed in northern Ecuador at the Bahia de Caraquez, Manibe Province, and from Tumaco, Colombia. TS was found in samples of moribund shrimp from Choluteca, Honduras, in January 1994 and from a shrimp farm in Kahuku, Oahu, Hawaii, in May 1994. Later in 1994 into early 1995, TS was diagnosed in shrimp sampled from disease outbreaks in ponds in Cartagena, Colombia, Guatemala, and Northern Brazil. Capture wild broodstock from El Salvador, which were shipped to an indoor reproduction facility in Florida, were reported to have come down with TS. In 1995, TS was identified in capture wild broodstock in a facility in southern Mexico and in farmed shrimp in various areas in Mexico. In May 1995, a TS outbreak started on shrimp farms in southern and central Texas resulting in an estimated $10 million in losses.

Up to August 1995, cases of TS had not been reported from shrimp farms in Panama, Costa Rica, Belize, Venezuela, Southern Brazil, and South Carolina. At present, TSV is believed to be eradicated from the facility in Florida.

Wild Shrimp Populations. Little is known about the presence and/or distribution of TSV in estuary and ocean fishery shrimp populations. TS has reportedly been observed in wild shrimp from the Gulf of Guayaquil, Ecuador. Apparently, further documentation is unavailable of TS from estuary or fishery shrimp populations in Central and South America. Several attempts to demonstrate TSV from capture wild broodstock, spawned eggs, or nauplii from El Salvador have produced negative results. Both enhancement and ISB methodologies were employed in these trials. To date, more than 150 randomly collected broodstock *P. vannamei* and offspring from more than 100 spawns have been tested with negative results for TSV. Although preliminary, results from these studies indicate that either TSV is absent from fishery populations of shrimp or its occurrence is very low or sporadic. Moreover, the supply of capture wild postlarvae and or gravid broodstock *P. vannamei* available to shrimp farmers has not shown a detectable decline in recent years, suggesting that, if TSV is present in these wild stocks, the impact of the infection is negligible. To date, the available information indicates TSV does not have a discernible impact to wild shrimp populations.

**Dissemination.** The geographic range of TS has expanded rapidly, and TS is now widespread in the Americas. From 1992 through early 1995, TSV moved from shrimp farms in the Gulf of Guayaquil, Ecuador, into neighboring farming regions and as far north as Texas. However, in general, the mechanisms for dissemination of TSV remain undocumented. Claims have been made that transfer of TSV has occurred with shipments of postlarvae and nauplii, but these assertions are unsupported by scientific data.

Limited data have shown TSV movement with shipments of contaminated broodstock *P. vannamei* from a facility in Kahuku, Oahu, Hawaii, into both Cartagena, Colombia, and an operation in Brazil. These shipments were done before it was known that TS was caused by a virus. However, retrospective studies demonstrated that TSV was present in broodstock that were examined at both shipment and receiving locals, clearly implicating the animals were incubating TSV prior to being transported. These observations reinforce the need for rapid methods of diagnosis and for the application of quarantine procedures for shipments at the port of entry.

Water boatmen *(Tricorixa reticulata)* collected from ponds with active TS have, in some cases, been shown by ISB tests to harbor TSV. Although it is easy to appreciate transfer of TSV between ponds on a farm by movement of water boatmen, the role these insects play as reservoir hosts for TSV or in dissemination of TSV over larger distances is uncertain.

Infective TSV has been demonstrated in seabird feces collected near ponds with TS. Waterbirds may be important as mechanical vectors of TSV.

Laboratory observations indicate that TSV survives a year or more in frozen shrimp tissues. Thus, movement of TSV between regions in frozen shrimp products should not be overlooked as a potential means for geographic transfer of this virus.

**Management Options.** From the perspective of the shrimp farmer, the most important aspect of TS is how to prevent or control the problem. Needless to say, the management of TS on shrimp farms has received intense interest and activity by shrimp farmers in regions that have been affected by this disease. Some management strategies being tried for TS control include (1) stocking of capture wild postlarvae; (2) use of *P. stylirostris;* (3) increased stocking density of postlarvae; (4) use of offspring spawned from *P. vannamei* that are TS survivors; and (5) maintenance of near-optimal water quality conditions in the growout ponds.

**Summary and Conclusions.** Mounting evidence indicates that TS is caused by a putative picornavirus called TSV. Descriptions are published of the disease signs and pathology of TS. Diagnosis of the acute phase of the disease is accomplished through demonstration of pathognomic lesions in the integument by application of standard histopathologic methods. Carrier state TSV infection is currently determined by use of a TSV-specific gene probe and/or specific-pathogen-free *P. vannamei* ISB.

Critical areas for research include studies to assess the benefit of stocking alternate species for growout on farms severely impacted by TS, development of *P. vannamei* that are less susceptible to mortality from TSV by selective breeding or immune enhancement, improving the understanding of the influence of environmental factors in the expression of TS, and molecular diagnostics for rapid, sensitive detection of TSV.

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(James A. Brock, DVM, Aquaculture Development Program, Department of Land and Natural Resources, State of Hawaii, 808–845–9561)

Questions about the Foreign Animal Disease Report may be sent to:

Dr. Sara Kaman, Editor
USDA, APHIS, VS
4700 River Road, Unit 40
Riverdale, MD 20737–1231

Thirty days before moving, send address changes and, if possible, mailing label from latest issue to:

Distribution Management Section
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